MicroRNA function is globally suppressed in mouse oocytes and early embryos.

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Public Summary:
Dicer, which is required for the processing of both microRNAs (miRNAs) and small interfering RNAs (siRNAs), is essential for oocyte maturation [1, 2]. Oocytes express both miRNAs and endogenous siRNAs (endo-siRNAs) [3, 4]. To determine whether the abnormalities in Dicer knockout oocytes during meiotic maturation are secondary to the loss of endo-siRNAs and/or miRNAs, we deleted Dgcr8, which encodes an RNA-binding protein specifically required for miRNA processing. In striking contrast to Dicer, Dgcr8-deficient oocytes matured normally and, when fertilized with wild-type sperm, produced healthy-appearing offspring, even though miRNA levels were reduced to similar levels as Dicer-deficient oocytes. Furthermore, the deletion of both maternal and zygotic Dgcr8 alleles did not impair preimplantation development, including the determination of the inner cell mass and trophectoderm. Most surprisingly, the mRNA profiles of wild-type and Dgcr8 null oocytes were essentially identical, whereas Dicer null oocytes showed hundreds of misregulated transcripts. These findings show that miRNA function is globally suppressed during oocyte maturation and preimplantation development and that endo-siRNAs, rather than miRNAs, underlie the Dicer knockout phenotype in oocytes.

Scientific Abstract:
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